We present a case of facet joint infection (pyogenic facetitis) due to *Eikenella corrodens*, diagnosed by physical examination, radiography, positive blood cultures, and response to antibiotic therapy. *E. corrodens* is a very rare cause of spine infection. There are fewer than 20 such cases reported in the literature, only one of which was diagnosed by non-invasive means, and none of which were isolated to the facet joint. We briefly review the microbiology of *E. corrodens* in addition to the diagnosis and management of spine infection.

**CASE PRESENTATION**

A 63-year-old woman, who was recovering from a lumbar strain incurred after she slipped and fell in a grocery store two months prior, noted increasing lower back pain for five days. On the day of admission, she experienced severe pain while attempting to stand from a seated position. She described the pain as sharp, centered just above her buttocks, non-radiating, exacerbated by walking, and relieved by lying on her side. She denied lower extremity weakness or paresthesia and had no loss of bowel or bladder function. She also denied subjective fevers, chills, or recent illness. Her back pain had become so severe that it prevented her from walking, and for this reason she reported to the emergency department (ED). Home medications for her lumbar strain included cyclobenzaprine, meloxicam, and tramadol. She has a 10-year history of mild osteoarthritis of her hands and knees for which she occasionally took acetaminophen. She took diltiazem, metoprolol, and hydrochlorothiazide for hypertension, and she had a brain aneurysm clipped in 1998. Her past medical, surgical, and family history was otherwise non-contributory, as was her review of systems.

On presentation, our patient had an oral temperature of 39.3 °C. She was in moderate distress secondary to back pain. Palpation of her L4 spinous process greatly exacerbated her pain, as did rolling from a decubitus to supine position. Her lumbar paraspinal muscles were tense but not appreciably tender to palpation. There was no erythema, warmth, induration, fluctuance, or integument defect overlying her lumbosacral spine. She had good rectal tone and no saddle anesthesia. Lower extremity tone, strength, and sensation were within normal limits. Straight leg raise did not elicit radicular pain, and she exhibited no meningismus. Her dentition was fair, without obvious caries, and no oral lesions were present. Cardiac examination revealed tachycardia but a regular rhythm without murmurs. She had no rashes.
or overt evidence of intravenous drug use such as tracking in her antecubital fossae or lesions on her extremities suggestive of skin popping.

Initial spinal radiography interpretation called attention to a grade 1 anterolisthesis of L3 over L4 without a pars defect, as well as degenerative changes in the lower thoracic spine. Vertebral heights and end plates were within normal limits, though there was a slight relative narrowing of the L3-L4 disc space (Figure 1). Contrast-enhanced computed tomography (CT) scan of the lumbar spine revealed neither a fluid collection nor obvious evidence of osteomyelitis. Magnetic resonance imaging (MRI) was contraindicated in our patient due to her brain aneurysm clip. White blood cell (WBC) count was $12.8 \times 10^3$ cells/μL [normal 4.5-11], with 86% neutrophils, 2% bands, 10% lymphocytes, and 2% monocytes. Erythrocyte sedimentation rate (ESR) was 20 mm/h [0-30], and C-reactive protein (CRP) was 10.4 mg/dL [0-0.9]. The neurosurgery service was consulted in the ED but found no indication for surgical intervention.

After obtaining blood cultures in the ED, we placed our patient on intravenous (IV) vancomycin and piperacillin-tazobactam. All four blood cultures (two aerobic and two anaerobic bottles) were reported as positive for gram-negative rods at 28 hours, and by culture day four, the organism was identified as *Eikenella corrodens*. As our hospital’s microbiology lab does not assess antibiotic sensitivities for this organism, we changed our patient’s regimen to ceftriaxone based on sensitivities reported in the literature. We chose a once-daily regimen in anticipation of continuing IV antibiotics in the outpatient setting. On further questioning, our patient denied recent dental work, ingestion of bones, instrumentation of her genitourinary or gastrointestinal tracts, human or animal bites, IV or percutaneous drug use, and licking her sewing needles.

Transthoracic and transesophageal echocardiograms on hospital days four and six, respectively, revealed mild mitral valve regurgitation but otherwise structurally normal valves with no evidence of vegetation or thrombus. On further review, the CT scan revealed inflammatory changes and bony destruction of the L3-L4 facet joints (Figure 2). Whole-body gallium scan, completed on day seven, supported these findings (Figure 3). As our patient had markedly improved, we did not pursue further tests such as a bone scan, CT-guided biopsy, or open surgical biopsy.

Several days after initiation of antibiotics, our patient tolerated lying flat on her back, was able to ambulate to the restroom with only mild discomfort, and her L4 spinous process had become less tender to palpation. By the time of discharge on hospital day nine, she was able to ambulate without pain, and her spine was tender only to deep palpation. Her oral temperature curve and laboratory measures had also improved (Figure 4). She was discharged on ceftriaxone, to be administered daily at our outpatient infusion center for a minimum of three additional weeks.

Our patient did not follow up in the infectious disease clinic as scheduled. We contacted her by phone on day 60, and she explained that home health administered her antibiotics for three weeks as ordered (four weeks in total) and then removed her percutaneous intravenous central catheter. She had no further back pain and felt very well. Prior to discontinuation of her antibiotics, the home health agency obtained a WBC count, ESR, and CRP – all of which had normalized.

**DISCUSSION**

*E. corrodens* is a fastidious, facultative anaerobic gram-negative rod. It is part of the normal flora in the human oral cavity, as well as on other mucosal surfaces such as the upper respiratory, gastrointestinal, and genitourinary tracts. Its genus namesake is the biologist Eiken, and its species name is derived from its tendency to create pits in agar growth medium. Historical but outmoded names include *Bacte-
Eikenella corrodens and HB-1. E. corrodens grows slowly in either aerobic or anaerobic environments, though 3-10% carbon dioxide enhances its growth. It is generally susceptible to B-lactam antibiotics, including penicillin, ampicillin, piperacillin, and both second- and third-generation cephalosporins; fluoroquinolones are also efficacious. It is resistant to methicillin and nafcillin, and some strains produce B-lactamases. E. corrodens is uniformly resistant to metronidazole and clindamycin and varies resistant to macrolides and aminoglycosides.

E. corrodens is of the HACEK grouping of organisms, the other members being Haemophilus aphrophilus, Actinobacillus actinomycetemcomitans, Cardiobacterium hominis, and Kingella kingae. HACEK organisms have in common gram-negative rod morphology, slow growth, in vitro growth enhancement by carbon dioxide, and a propensity to infect heart valves. Infections due to HACEK organisms are typically indolent, with one week or longer between inoculation and the onset of symptoms. Such infections may likewise require protracted courses of antibiotics to achieve cure. Many infections with E. corrodens are of the head and neck, due to high bacterial load within the oral cavity and subsequent direct invasion via mucosal defects, particularly in the setting of oropharyngeal cancer. Skin infections usually result from direct inoculation of oral secretions such as occurs with human bites or closed-fist injuries incurred by striking someone in the mouth. Another mechanism of such infections is injection of drugs subdermally (skin popping) after licking the needle. More distant infections are due to hematologic spread, and some cases have been attributed to transient bacteremia following dental work or associated with overtly compromised dentition. However, the source of bacteremia often cannot be determined with certainty.

Our patient developed an infection in her facet joint (pyogenic facetitis) due to hematogenous dissemination of E. corrodens. Data leading to this diagnosis includes our patient’s clinical presentation, findings on physical examination, radiographic evidence, bacteremia, and both clinical and laboratory response to antibiotics. MRI would have provided additional useful data, but it was contraindicated for our patient. Confirmation of the diagnosis via tissue biopsy and culture was not pursued as it would have incurred significant risk without clinical benefit. The first documented case of a spine infection due to E. corrodens was diagnosed in 1979 using similar clinical criteria as we used here, though the first case was of osteomyelitis rather than facetitis and thus, more amenable to radiographic diagnosis.4 A large multicenter study found that E. corrodens bloodstream infection was quite uncommon, with only 4 (0.007%) of 59,203 blood cultures positive for the organism.

This same study showed that blood cultures do not have to be held longer than the usual five-day period for the detection of HACEK organisms.

A PubMed search was conducted using the following search terms: (“Eikenella corrodens” OR “Bacteroides corrodens” OR “HB1”) and (“vertebral osteomyelitis” OR “spondylitis” OR “discitis” OR “vertebra” OR “facet joint” OR “spine”). This search and subsequent bibliographic review yielded 15 cases of E. corrodens spine infection. Nine cases were attributed to hematologic spread.6-13 The remaining six cases were attributed to direct inoculation of the vertebral or paravertebral space via surgery or fish bones penetrating the esophagus.14-16 Unlike most reported cases, our patient had no predisposing factors such as malignancy, diabetes, dental procedures, vascular instrumentation, or direct exposure of the spinal column to the environment. Ours is also the first reported case in which a facet joint is identified as the locus of infection.

Facet joints are infrequently reported as loci for spine infection, though there is some suggestion this is more common than previously suspected. Clinical symptoms and signs are very similar to vertebral osteomyelitis, for which there is much more data. In contrast to vertebral osteomyelitis, facetitis generally has an onset which is much more acute, perhaps driven by a more focal sensation of pain and marked muscle spasm. Facetitis has a stronger predisposition for the lumbar spine than osteomyelitis, and it is also more commonly associated with fever. Unlike vertebral osteomyelitis, in which joint space narrowing and endplate destruction is typically evident at the time of patient presentation, plain radiography is generally much less useful for the diagnosis of facetitis. Staphylococcus aureus is an even more dominant etiologic agent of facetitis, while gram-negative organisms such as Escherichia coli are very rarely implicated in this disease.20

Vertebral osteomyelitis, the most commonly encountered type of pyogenic spine infection, has an incidence of 2.4 cases per 100,000 persons, with the highest prevalence in...
older men.21 While back pain is the most common presenting symptom (86%), pain on spinal percussion is relatively insensitive in the absence of a co-existing epidural abscess. Fever is also unreliable, perhaps due to the often coincident use of non-steroidal anti-inflammatory agents or acetaminophen.21,22 Lower segments of the spine are more commonly affected: lumbar (58%), thoracic (30%), and cervical (11%).22 While leukocytosis has poor sensitivity, that of the non-specific inflammatory markers ESR and CRP approaches 100%.21 Blood cultures unfortunately have a sensitivity of only 58%, but when positive in the presence of compatible clinical findings, they may obviate the need for invasive testing such as a needle or open biopsy.22 Unless there is an associated abscess requiring drainage, vertebral osteomyelitis can be successfully treated with antibiotics alone. The most common pathogen is *S. aureus*, followed distantly by *E. coli*. Less commonly, in the presence of foreign bodies, low-virulence organisms have been reported as causal.21 There are no official guidelines for the management of vertebral osteomyelitis, though the Infectious Diseases Society of America (IDSA) plans to release guidelines in 2013. Current management is based on expert opinion and, as in our case, *E. corrodens* antibiotic susceptibilities reported in the literature.

**CONCLUSIONS**

*Eikenella corrodens* is rarely isolated in the blood and is an unlikely cause of spine infection. This case is a reminder that normally commensal organisms, given the opportunity, can cause significant disease even in healthy individuals. It also demonstrates that invasive procedures are not always essential in the diagnosis and management of spine infection, even if that infection subtly affects the facet joints. Facetitis is an important cause of acute lower back pain, and prompt recognition and treatment can prevent significant morbidity.

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